

REFERENCES

- Bartlett JG. Preventative treatment in human immunodeficiency virus infection: vaccinations. *Infect Dis Clin Prac* 1996; 5:25–31
- Kroon FP, van Furth R, Bruisten SM. Effects of immunization in human immunodeficiency virus type 1 infection. *N Engl J Med* 1996; 335:817–822
- Toerner JG, Mathews WC. Guidelines for immunizations in HIV-infected patients. *Immunol Allergy Clin N Am* 1997; 17:195–205

Drug Reactions in HIV/AIDS

Adverse reactions to drugs are frequent in patients with human immunodeficiency virus (HIV) infection and AIDS. The most commonly prescribed drug for AIDS patients is trimethoprim-sulfamethoxazole (TMP-SMX); it is also the most common source of adverse reactions. Up to 40% of patients receiving high-dose TMP-SMX develop a maculopapular diffuse rash, often with fever and malaise. Similar reactions have been reported with clindamycin, diapsone, pyrimethamine/sulfadoxine, aminopenicillins, clavulanate, thalidomide, atovaquone, nevirapine, delavirdine, rifampin, probenecid, and 1592 (a new antiviral drug).

The cause of these reactions is not clear. Immunologic processes might be involved, since patients with HIV and AIDS demonstrate immunoactivation as well as immunodeficiency. A polyclonal increase of immunoglobulins (including IgE), increased circulating immune complexes, and an increased number of activated CD8⁺ cells may be secondary to various infections. Abnormal hepatic metabolism may result in the persistence of drug metabolites that act as antigens or have direct cellular toxicity. Alternatively, an incompetent immune system may fail to clear the metabolites. Finally, incidence of certain viral infections, such as herpes simplex virus, hepatitis B virus, cytomegalovirus, and Epstein-Barr virus (EBV), is increased in patients with HIV and AIDS. These infections may stimulate the immune system, predisposing the patients to drug reactions such as that between EBV and aminopenicillins. There is no conclusive evidence that the incidence of IgE-mediated reactions to drugs, such as penicillin-induced anaphylaxis, is increased in patients with HIV/AIDS. Rather, the incidence of some adverse drug reactions seems to increase with advancing immunodeficiency. A possible participation of hepatic drug metabolism may explain the success of incremental dosing (as with nevirapine and delavirdine) and desensitization protocols in minimizing the problems.

The most common clinical presentation of an adverse drug reaction in patients with HIV/AIDS is rash. The rash, which starts 7 to 12 days after initial exposure to the culprit agent, is usually diffuse, erythematous, maculopapular, and pruritic and is frequently accompanied by fever and malaise. Conjunctivitis is common, but mucous membrane involvement is not. Susceptible patients may have identical or nearly identical symptoms from several agents. More severe reactions, such as Stevens-Johnson syndrome, bullous lesions, and toxic

epidermal necrolysis, also may occur. Hepatic and hematologic abnormalities can stem from immune-mediated injury or direct toxicity; rare cases have been reported of eosinophilic pneumonia possibly secondary to drugs.

The diagnosis of adverse drug reactions rests mainly on a good history, especially a detailed drug history, timing of introduction of the suspected agent, and clinical symptoms and physical examination.

Withdrawal of the offending agent is usually successful in treating the reactions. Although severe bullous rashes are often treated with corticosteroids, the effects of this treatment are difficult to assess. We have noted remarkable responses to intravenous immunoglobulin infusion in a few seriously ill patients with bullous skin rashes.

Although avoiding the offending agent is the safest method to treat the problem, it may not be the best approach if the agent is the drug of choice and there is no tolerated alternative. In such circumstances, a careful desensitization may be attempted. Desensitization has been successful with TMP/SMX, clindamycin, sulfadiazine, and other agents, but probably should not be attempted for such serious or life-threatening reactions as anaphylaxis, bullous dermatitis, or renal or hepatic failure.

HAMID HUSSAIN, MD
GILDON BEALL, MD
MARGERY SANWO, MD
Torrance, California

REFERENCES

- Beall G, Sanwo M, Hussain H. Drug reactions and desensitization in AIDS. *Immunol Allergy Clin N Am* 1997; 17:319–337
- Sanwo NC, Nwadiuko R, Beall GN. IVIG in severe drug reactions. *J Allergy Clin Immunol* 1997; 99:1112–1115

Latex Allergy—The Latest Insights

Urticaria to latex has been reported as early as 1927 and more recently in 1979. Fatal reactions to latex-tipped barium enema catheters brought attention to the potentially serious nature of the allergy around 1990. In recent years, awareness of the condition has risen, as has exposure to latex: more than 40,000 products contain latex, and latex gloves are omnipresent in health care settings. As a result, more and more latex-sensitive patients are being identified. Groups reported to have high prevalence rates of latex allergy include health care workers, dental workers, rubber industry workers, housekeepers, spina bifida patients, and patients undergoing frequent catheterizations or multiple surgeries.

Two potential mechanisms for sensitization are common: direct contact and inhalation of aerosol latex proteins carried on the cornstarch of powdered gloves. Latex-sensitive individuals experience symptoms that vary from localized hand symptoms such as itching or urticaria to a wide range of systemic reactions including